PATHOGENIC ROLE FOR ARGENINE VASOPRESSIN (AVP) AND CATECHOLAMINES (EP & NEP) IN VASOVAGAL SYNCOPE

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Elevation of EP, NEP and AVP have been seen in vasovagal syncope (VVS). AVP may 'sensitise' left ventricular (LV) receptors, and EP may stimulate them. To elucidate timing of hormone changes in VVS, blood was sampled for EP, NEP and AVP during prolonged 60° head-up tilt (HUT) in 16 patients (pts) with unexplained syncope (UES) with previous tilt induced vasovagal syncope and two non-syncopal controls (C) asymptomatic during prior tilt.

RESULTS:

	Syncopal (14 pts)			
Time	EP	NEP	AVP	EP =pmol/L
B/line	0.01±0.02	0.4±0.04	2.8±0.4	NEP=pmol/L
T+0 min	0.12±0.02	0.5±0.07	4.0±1.1	AVP=pmol/L
T+10 min	0.15±0.02	0.7±0.07	20±4.2	
Sync	0.84±0.27	0.7±0.06	21±3	
Recvy	0.37±0.1	0.6±0.07	14±2.8	
	Non-syncopal (2 pts, 2 C)			
B/line	0.05±0.01	0.35±0.15	0.8±0.25	
T+0 min	0.06±0.03	0.56±0.15	1.4±0.25	
T+10 min	0.09±0.04	0.65±0.17	2.3±0.4	
T+20 min	0.12±0.04	0.6 ±0.1	1.9±0.4	
T+40 min	0.17±0.11	0.53±0.1	2.1±0.44	
T+60 min	0.15±0.07	0.56±0.1	2.2±0.46	

At baseline supine ($p<0.01$) at 10 mins into HUT ($p<0.01$) and as syncope was evolving ($p<0.01$), AVP was higher in those with syncope than those without. EP was also higher in syncopal pts ($p<0.05$). There was no difference in NEP profiles.

CONCLUSION: Susceptible pts during HUT have marked elevation of AVP before and during tilt induced VVS which may 'sensitise' LV receptors making them more sensitive to high EP levels and result in VVS.

ECHOCARDIOGRAPHIC EVIDENCE OF LEFT VENTRICLE HYPERCONTRACTILITY DURING HEAD-UP TILT IN PATIENTS WITH NEUROCARDIOGENIC SYNCOPE

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It has been suggested that in Pts with neurocardiogenic syncope (NCS) exagerated left ventricular contractility may play a role in vasodepressor reflex activation resulting in arterial hypotension and ultimately syncope. Head up tilt (HUT) test is currently utilized to evaluate PTs hemodynamic response to upright position. However, no data exists to evaluate LV dimensions and function during HUT. LV fractional shortening (LVFS), ejection fraction (LVEF) and end systolic volume (ESV) were compared during the HUT in Pts with NCS (Group I, 7 Pts), unexplained syncope and negative HUT (Group II, 4 Pts) and controls (Group III, 11 Pts).

GROUP	I	II	III	P VALUE I vs II, III
LVFS%	45.5±7.4	31.2±3.3	35.4±7.2	p<0.05
LVEF%	81±6.4	76±6.5	70.7±2.3	p<0.001
ESV	3.2±0.9	3.1±0.7	5.2±1.9	p<0.05

These significant changes in LV parameters consistently preceded the onset of hypotension. Thus, in Pts with unexplained syncope, LV hypercontractility, reduced end systolic volume, and increased LVEF during HUT are unique findings occurring in Pts with NCS and may have an important pathophysiologic role.

INVESTIGATION OF THE MECHANISM OF SYNCOPE IN PATIENTS WITH HYPERTROPHIC CARDIOMYOPATHY.

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Syncope occurs in 10-20% of patients with hypertrophic cardiomyopathy and is a difficult management problem. There are numerous potential arrhythmia and hemodynamic causes. To evaluate the utility of ECG monitoring, exercise and electrophysiologic testing, 16 consecutive pts presenting with syncope and 16 with refractory symptoms were investigated. All 32 pts had 2D echo/Doppler, 48 hour ambulatory ECG, maximal exercise testing with direct measurement of arterial pressure and EPS including assessment of sinus node, AV node and His Purkinje function and RV stimulation (Wellens protocol stage 8). When a cause of syncope was not established, repeated 48 hour ECG monitoring and/or patient activated recorders were used. Pts with syncope had LV wall thickness (1.6-3.8, mean 2.5 cm), LV end diastolic and end systolic dimensions (3.0-5.0, mean 4.5 cm and 1.8-3.6, mean 2.6 cm) and LA dimension (3.0-6.0, mean 4.4 cm) similar to pts without syncope. Three of 16 (19%) with syncope had a resting LV gradient >20 mmHg. A likely cause of syncope was established in 9 of 16 (56%): documented at the event in 2 (1 ventricular fibrillation while walking, 1 sustained monomorphic ventricular tachycardia (SMVT)); during initial (1 pt) or prolonged (3 pts) ECG monitoring (3 paroxysmal atrial fibrillation (PAF), 1 SMVT); during exercise testing in 1 (ventricular fibrillation after 2 minutes of recovery) and during electrophysiological study (EPS) in 2 (1 PAF with hypotension, 1 SMVT). Exercise hypotension (fall of >20 mmHg from peak pressure) developed in 6 pts with and 9 without syncope and was not associated with symptoms of impaired consciousness. Sustained arrhythmias were not documented in pts without syncope. In summary: 1) supraventricular arrhythmias were the commonest cause of syncope; 2) conventional assessment documented a likely cause of syncope in 56%; 3) initial ECG monitoring, exercise testing and EPS were useful, but repeated ECG monitoring/patient activated recording was the most useful investigation.

DYNAMICS OF ATRIOVENTRICULAR NODAL CONDUCTION RATIOS OF UNSTABLE 2:1 BLOCK PRODUCED BY ATRIAL PACING.

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For the purposes of this study, episodes of unstable 2:1 AV nodal block were defined as ≥ 1 runs of 2:1 block initiated by (thus including) a run of 3:2 block and terminated when another episode started. Episodes of unstable 2:1 AV nodal block were found, during incremental atrial stimulation, in 9/67 (13.4%) of retrospectively reviewed charts of patients referred for diverse supraventricular arrhythmias. They always occurred at cycle lengths between those at which Wenckebach block and stable 2:1 block appeared. Longest cycle length of Wenckebach block (range = 400-250 ms; mean = 310 ms) exceeded, in every case, longest cycle length of stable 2:1 block (range = 350-200 ms; mean = 256 ms). Unstable 2:1 AV nodal block cycle length values were in between (range = 350-220 ms; mean = 276 ms). The AV nodal conduction ratios (during 1:1 block referred as M:N) at which episodes of unstable 2:1 AV nodal block occurred were (with numbers of episodes in parenthesis): 5:3 (15); 7:4 (14); 9:5 (7); 11.6 (1); and 15.8 (2). This occurred because each episode was composed of a 3(M):2(N) ratio plus $\geq$ 2(M):1(N) ratios (thus conforming with a 2N-1:N ratio). In conclusion, these episodes of unstable 2:1 AV nodal block had rich, heretofore undescribed, dynamics characterized by a universal 2N-1:N AV nodal conduction ratio, which invariably had a first number that was odd.